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Transfusion malaria in developing countries

Transfusion malaria remains a problem in developing countries, and Edrissian[1] has reviewed 111 cases of transfusion malaria recorded in Iran from 1963 to 1972, while Wickramasinghe[2] has recently dealt with 16 cases of accidental transfusion malaria that occurred in Sri Lanka. Cases have been reported from a large number of countries, some of the early accounts having come from China.[3] There is little doubt, however, that transfusion malaria is still grossly underreported.

The incubation period of blood-induced infection is quite different from that of mosquito-transmitted malaria, since in the former there are no tissue stages in the liver. Both the interval between infection and the appearance of parasites in the blood—the prepatent period—and the presymptomatic period depend on four factors: the number of parasites introduced; the method of inoculation; the susceptibility of the recipient; and the length of blood storage before transfusion. Reviewing a series of over 100 cases of transfusion malaria in Rumania, Lupascu *et al*[4] pointed out that 86% of all cases occurred when the blood was stored for less than five days; cases after 7-12 days were rare, while infections with the blood stored for 13 or more days were exceptional. In addition to infection from blood transfusion accidental transmission of malaria may also occur with plasma if some stray red blood cells happen to contain plasmodia[5] or as a result of leucocyte transfer.[6] Most reported cases of transfusion malaria are due to *Plasmodium malariae*, followed by *P vivax* and *P falciparum*.

Neither systematic screening—with various serological techniques—nor premedication of suspected donors is a practical solution in developing countries. The generally accepted procedure in areas where non-immune persons may receive blood that may possibly contain scanty malaria parasites is the prophylactic administration of antimalarial drugs to the recipients.[7 8] As there is no likelihood of true relapses occurring after blood transmission of *P vivax*, *P malariae*, or *P ovale*, radical treatment with an 8-aminoquinoline such as primaquine is not required. A single dose of 600 mg base chloroquine to the recipient 24 hours before or on the day of transfusion seems to protect from induced malaria. It may be prudent, however, not to rely on a single dose of chloroquine but to give all non-immune recipients the standard three-day course of antimalarial treatment.[9 10]

In parts of the world where strains resistant to the 4-aminoquinolines are known to occur, such as SE Asia and S America, the preventive administration of sulfadoxine, 1·5 g, together with pyrimethamine, 75 mg (Fansidar), is advisable. Once again it may be prudent to give a full three-day course of curative treatment—quinine (four doses, 540 mg each dose, given at intervals of 12 hrs) and then a single dose of sulfadoxine-pyrimethamine.[11] A detailed account of the problem of blood transfusion and the whole range of tropical diseases (including malaria) is available in the comprehensive review by Bruce-Chwatt.[12]

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Screening for presymptomatic coronary disease

Coronary artery disease has been shown to be the cause of death in four out of every 10 men in studies with high necropsy rates.[1] Yet while clinical procedures and simple laboratory tests permit recognition of traits associated with increased risk of coronary disease,[2] they may not identify its presence in any one individual—let alone whether it represents a danger to him (or to others). One American man in five[3] may expect to have symptoms from a coronary lesion by age 60, but not all of those with symptoms of coronary disease will die from it, and half of those who do will never have had recognised symptoms. In these cases of unexpected sudden death there is commonly serious obstructions of two or three major coronary arteries,[4] so that it follows that angina is both a poor guide to risk and an insensitive marker of underlying coronary obstructive disease. Follow-up studies after coronary angiography have shown that risk of death is closely related to the severity of coronary obstruction,[5 6] and that the 25% or so patients with clinically diagnosed angina but normal coronary arteries[7] have a normal (or better than normal) prognosis.[6]

Most coronary deaths are due to sudden ventricular fibrillation without infarction or occur within an hour of onset of infarction,[8] but even myocardial infarction may not be a clinical event. The Framingham study showed that one-quarter of the individuals who developed electrocardiographic evidence of myocardial infarction had not been recognised as having had an infarct by their doctors.[9]

Short of coronary angiography, then, recognising coronary disease is not always possible. An abnormal electrocardiogram may falsely suggest ischaemia, and one in four ECGs reverts to normal after actual infarction.[9] Nevertheless, certain ECG abnormalities are associated with a high incidence of coronary disease. Most important of these is left ventricular hypertrophy,[10] but other pointers include intraventricular conduction defects and non-specific ST and T wave changes. Changes in the ECG during or after effort lack specificity[11] if an ST segment depression of 0·5 mm is accepted, and show inadequate sensitivity if the measure is set at 2 mm: at present 1 mm is the usual criterion. Unfortunately, the exercise ECG also suffers from lack of reproducibility: tests may vary between